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EXAMINER	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/789,051	KRIEG ET AL.	
	Examiner	Art Unit	
	OLUWATOSIN OGUNBIYI	1645	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *23 February 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *28,31-33,35,37,39,40 and 42-47* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *28, 31-33, 35, 37, 39, 40 and 42-47* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

Response to Amendment

The amendment filed 2/23/09 has been entered into the record. Claims 28, 31-33, 35, 37, 39, 40 and 42-47 are pending in the application and under examination

Rejections Withdrawn

The rejection of claims 28, 31-33, 35, 37, 39, 40, 42, 43-46 provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claim 36 of copending Application No. 10/787,737 is withdrawn in view of abandonment of the '737 application.

The rejection of claims 28, 31-33, 35, 37, 39, 40 and 42-47 under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement is withdrawn in view of the amendment to the claims.

Rejections Maintained

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

The rejection of claims 28, 31-33, 35, 37, 39, 40 and 42-47 rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement is maintained for reasons made of record in the previous office actions filed 3/22/07 and 1/18/2008 and 8/26/08 to the extent they apply to the instant claims as amended and as set forth below.

The claims are drawn to a method for treating or preventing an immune system deficiency, comprising administering to a subject an oligonucleotide containing an unmethylated cytosine-guanine to treat the immune system deficiency, wherein the oligonucleotide is stabilized, wherein the oligonucleotide comprises 5'-TGACGTT-3' wherein the oligonucleotide comprises a phosphorothioate modification wherein the subject has a cancer, viral infection or a bacterial infection.

The specification defines immune system deficiency as follows:

A disease or disorder in which the subject's immune system is not functioning in normal capacity ***or*** in which it would be useful to boost a subject's immune response for example to eliminate a tumor or cancer (e.g. tumors of the brain, lung (e.g. small cell and non-small' cell), ovary breast, prostate, colon as well as carcinomas and sarcomas) or viral (e.g. HIV, herpes), fungal (e.g. Candida sp.), bacterial or parasitic (e.g. Leishmania, Toxoplasma) infection in a subject.

See p. 11 lines 21 to 26 of the specification.

Applicants in their arguments specify that they are claiming the latter type of immune system deficiency and that the claims do not encompass the treatment of a disorder in which the immune system is not functioning in a normal capacity such as the disorders listed on pages 3-4 of the Office action mailed 8/26/08 and that the claims have been amended to clarify this. See Applicants arguments p. 5 in the reply filed 2/23/09.

Thus, the nature/scope of the instant invention is drawn to the boosting of a subject's immune response to treat or eliminate any type of cancer, any type of viral infection or any type of bacterial infection.

The nature/scope of the instant invention also includes preventing diseases or disorder due to any type of cancer, any type of viral infection and any type of bacterial infection in a subject having the cancer, viral or bacterial infection by treating the cancer or bacteria or viral infection, as the claim also requires "preventing an immune system deficiency" in addition to "treating an immune system deficiency".

The scope of 'subject' set forth includes humans, dog, cat, horse, cow, sheep, goat, chicken, monkey, rat, mouse etc. See specification p. 11 last two lines

The Breadth of the Claims

The scope of bacterial infections to be treated is extremely broad. The scope of bacterial infection includes a plethora of pathogenic bacteria that are morphologically distinct having different characteristics. The mechanisms of infection and pathogenesis of the diverse bacteria is also distinct (see Medical Microbiology 4th ed. 2002 Chapter 19 p. 176-184 and Chapter 45 p. 421-426). Examples of infection causing bacteria to name a few include *Mycoplasma sp., Staphylococcus sp., Streptococci sp., E.coli, Mycobacterium sp., Borrelia sp., Clostridium sp., Chlamydia sp.* etc.

The scope of cancers to be treated or prevented is also broad. There are many different types of cancers affecting different organs or tissues of the body e.g. ovary, breast, skin brain, prostate, colon etc.

The scope of viral infections to be treated is also broad. Viral infections include HIV, herpes, human papillomavirus, influenza virus, yellow fever (flavivirus), SARS (coronavirus), cytomegaloviruses etc.

Guidance in the specification/ Presence or absence of working examples/ State of the Prior Art/ predictability or unpredictability of the art

The teachings of the specification are limited to *in vitro* and *in vivo* data that demonstrate that unmethylated cytosine –guanine containing oligonucleotides stimulates B-cells and induces the production of cytokines and data that demonstrates induction of IL-6 in mice injected with said oligonucleotides. The specification at the time of filing does not correlate the immune responses generated by administering an oligonucleotide containing an unmethylated wherein the oligonucleotide comprises 5'-TGACGTT-3' wherein the oligonucleotide comprises a phosphorothioate modification *in vitro* with treating the breadth of cancers, viral infections or bacterial infections.

Applicants argue that the fundamental invention is directed to a class of molecules useful in treatment of diseases such as cancer and infection disease and is based at least in part on the discovery that the immune system detects bacterial DNA by the presence of unmethylated CpG nucleotides and the immune response generated is predictive of the utility of the class of molecules in treatment of cancer and infectious diseases. Applicants argue that that one aspect of

the invention is the recognition that the same type of immune response triggered through this defense pathway (immune response to bacterial DNA) can be directed against a cancer or viral or bacterial infections and that clinical trials involving administration of bacterial DNA to humans demonstrated positive effects on cancer patients.

Applicants' arguments are carefully considered but are not persuasive. While at the time of the effective filing date of the instant application (1994), bacterial DNA comprising CpG motifs was indeed known to be immunostimulatory, mere immune response does not predict the efficacy of the instant oligonucleotide containing an unmethylated wherein the oligonucleotide comprises 5'-TGACGTT-3' wherein the oligonucleotide comprises a phosphorothioate modification in treating cancers, viral infections/diseases or bacterial infections/diseases.

Treatment of cancer, viral infections or bacterial infections is very complex and dependent on many factors. As mentioned above the induction of an immune response in combating cancer or bacterial or viral infections is not a simple matter.

Even though the specification demonstrate that unmethylated cytosine –guanine containing oligonucleotides stimulate B-cells and induce the production of Il-6, IgM and induction of natural killer cell activity, however, efficacy of recombinant IL-6 in treating one type of intracellular bacterial infection in mice was time dependent and was not effective when administered 24hr post infection but effective at 4hr post infection. This demonstrates the role of IL-6 in the early response to bacterial infection (Liu et al. Infection and Immunity, Oct. 1992, p.4402-4406). Therefore, the time of administration of said oligonucleotide is critical for the innate response to have an effect. Also said oligonucleotide may be more efficacious when administered before bacterial or viral infection as demonstrated by Liu et al before the bacteria have the chance to evade the immune system. The specification has not addressed this issue by comparing the efficacy of said oligonucleotide when administered before infection or administered after infection.

Further as to treatment or preventing cancers in a subject who has a cancer, Kataoka et al (Jpn. J. Cancer Res vol. 83 p.244-247, 1992, cited in IDS) show anti-tumor activity of synthetic oligonucleotides containing cytosine-guanine in a murine tumor system with sequences from cDNA encoding proteins of *Mycobacterium bovis* BCG. Said anti-tumor activity correlated with NK cell activity and interferon inducing activities. However, Kataoka et al does not disclose an

oligonucleotide containing an unmethylated wherein the oligonucleotide comprises 5'-TGACGTT-3' wherein the oligonucleotide comprises a phosphorothioate modification. Furthermore, Kataoka et al teaches that the efficacy of the BCG bacterial DNA derived oligonucleotides used was not only dependent on the presence of the CG motif but the sequence context of the CG motif was important i.e. mere presence of the CG motif does not predict efficacy. The active oligonucleotides contained a hexameric palindromic sequence that contained the CG motif (see Kataoka et al p. 245) and Kataoka et al concluded that particular hexameric structures are essential for expressing the biological activities of oligonucleotides and for antitumor activity. Thus, contrary to Applicants arguments, the mere presence of the CpG dinucleotide does not predict the efficacy in treatment of any type of cancer or even bacterial or viral infections.

Even after filing reference disclosing the use of a CpG oligonucleotide comprising the instant 5'-TGACGTT-3' (submitted in reply to the Office action dated 1/8/08 –see Applicants arguments of 7/18/08) demonstrates all the complexity involved in treating one type of cancer in a mice model. Sfondrini et al was considered and analyzed in detail in the previous Office action mailed 8/26/08, see p. 8-10. As set forth previously, Sfondrini et al (FASEB 2002 vol. 16 p. 1749-1754) provided by Applicants teaches an oligonucleotide ODN 1668 that includes TGACGTT. Sfondrini et al does teach that phosphorothioate modified ODN 1668 prevented the development of spontaneous mammary tumors in 4 out of 11 mice after 380 days (FVB-NeuN transgenic mice were treated i.p. with CpG ODN every 10 days starting at 10 wk of age) while all untreated mice developed mammary tumors before 305 days of age (p. 1750 under oligonucleotides and under results and p. 1751 fig. 1). However, the results of Sfondrini are limited to treatment or prevention of one type of (cancer mammary adenocarcinoma tumors) in a murine subject while the instant claims are partially drawn to treating or preventing any type of cancer in a subject including humans who has cancer. Applicants submit that at the time the priority patent application was filed it was known in the art that induction of interferon gamma, Il-12, IL-6 as well as NK cell activation was useful in the treatment of cancer. However, Brunda et al (J. Exp. Med vol. 178, Oct 1993, p. 1223-1230) teaches the differences in responses to IL-12 stimulation of human versus murine NK or T cells in vitro in that said human cells secrete tumor necrosis factor while said murine T cells do not produce TNF (see p. 1228 column 1 last

paragraph). Thus, there are differences in the response to Il-12 between human and murine NK or T cells and Brunda et al (J. Exp. Med vol. 178, Oct 1993, p. 1223-1230) teaches that more work needs to be done to determine if the activity demonstrated in animals can be translated into efficacy against human malignancies (p. 1228 column 2 last paragraph). The unpredictability of using results in mice to predict efficacy in humans is even set forth post filing- Krieg et al (Proc Am Thorac Soc vol. 4 p. 289-294, 2007, see p. 289 left column under *the role of TLR9 in the mechanism of action of CpG ODNs*) teaches that it is difficult at best to use observations with CpG ODNs in murine studies to predict accurately the effects of TLR9 (i.e. the cellular receptor for CpG ODN) activation in humans because the cellular patterns of TLR expression vary between species so the results of TLR stimulation (in mice, for example) may not be predictive of what will occur in another (humans).

In addition, conditions for treating the mice already having the mammary adenocarcinoma tumors varied (see Sfondrini et al). In mice bearing small spontaneous mammary tumors, no significant tumor inhibition was observed using increased i.p. doses and increased frequency of administration whereas significant inhibition was observed when 40 ug of the CpG ODN were injected at the tumor site for 5 days (p. 1751 column 2 first complete paragraph). Sfondrini et al further demonstrates the complexity of treating the tumors in mice. Mice inoculated i.v. with N202.1A carcinoma cells formed significantly fewer lung metastases after 4 wk if treated with CpG ODNs (20 ug/mouse) 4h before or 2h after tumor cell inoculation compared with control and mice administered 40 ug CpG ODN administered i.p. 4 hours before and in the 4 subsequent days had inhibition of experimental metastases although still incomplete. No inhibitory effect was observed when CpG-ODN was administered 48 h after N202.1A cell injection. Thus, even in mice, the treatment of one type of cancer i.e. mammary adenocarcinoma with phosphorothioate CpG ODN comprising TGACGTT is complex and is dose, route and schedule of treatment dependent.

One of skill in the art at the time of filing (1994) could not predict the efficacy of one type of phosphorothioate modified unmethylated cytosine guanine containing oligonucleotide comprising 5'-tgacgtt- 3'for the treatment of all types of cancer in subjects having cancer. Hiyashi et al (Proc. Japan Acad., 70, Series B (1994) 205-209) demonstrates that different types of cancers at different stages of progression respond differently to treatment. Applicants submit

that Hayashi et al teaches the importance of interferon(IFN)-gamma in treatment of cancer. However, in Hiyashi et al (Proc. Japan Acad., 70, Series B (1994) 205-209) while the BCG cell wall skeleton treatment induced detectable levels of IFN-gamma in all surviving patients as compared to those who did not survive their cancer, the reference is drawn to treatment of cancer patients with BCG-Cell Wall Skeleton not to the treatment with CpG oligonucleotides. Furthermore, not all cancer patients produced IFN-gamma in response to the BCG cell wall and the different responses may be due to the stage of cancer, type of cancer and initial treatment (surgical operation and/or radiation or chemotherapy, see table 1 p. 207). Thus, the efficacy of the BCG cell wall in treating these patients was dependent on many factors and not merely production of IFN gamma – not all the patients were able to produce IFN-gamma in response to BCG cell wall. Also, it is impossible to predict from Hiyashi et al who used BCG cell wall, whether subjects with different cancers and/or at different stages of other types of treatment and/or at different stages of cancer would respond to the instant oligonucleotide to produce the cytokines that can treat said cancer.

Applicants cite U. S. Patent No. 4,883,662 (Nov. 28, 1989) to Stout for the teaching that increasing NK cells in the blood of cancer patients should be an advantage in cancer treatment (summary of the invention) because NK cells have known activity against tumor cells (abstract). The patent in Example II teaches the administration of a parvovirus immunized positive serum to a terminally ill cancer patient and there is no teaching of administration of CpG oligonucleotides. Furthermore, the patients condition deteriorated during therapy and at the end of the therapy had noted no unusual qualitative differences during the treatment period. Furthermore the patient finally died even though the serum increased the NK cell population in that patient. The Stout patent demonstrates that mere increase of NK cells in a cancer patient does not predict that sad cancer is treated or new tumors are prevented.

Applicants' submission that one skilled in the art would recognize the utility of treating cancer and infection based on the disclosure and data provided in the instant patent application is carefully considered. However, as mentioned previously the data provided in the specification is directed to *in vitro* and *in vivo* data that demonstrate that unmethylated cytosine –guanine containing oligonucleotides stimulate B-cells and induce the production of cytokines and in vivo data that demonstrates in vivo induction of IL-6 in mice injected with said oligonucleotides. The

specification is devoid of any data correlating the immune responses generated with treatment of any type of cancer or any type of bacterial or viral infection in any subject. The fact that an applicant has disclosed a specific utility for an invention and provided a credible basis supporting that specific utility does not provide a basis for concluding that the claims comply with all the requirements of 35 U.S.C. 112, first paragraph (MPEP 2107.01. General Principles Governing Utility Rejections).

Applicants submit that at the time the priority patent application was filed it was known in the art that induction of interferon gamma, Il-12, IL-6 as well as NK cell activation was useful in the treatment of cancer.

Applicants submit that Trinchieri et al. Blood, v. 84, December 15, 1994 teaches:

> Page 4021 describes the role of IL-12 in anti-tumor immunity. Specifically, it is taught that "studies using transplantable tumors in experimental animals have shown a dramatic affect of IL-12 in decreasing tumor growth and metastasis formation and in significantly delaying death. Systemic Daily Treatment (5 days per week) had a significant inhibitory affect on the growth of metastasis induced by intravenous injection of B 16 melanoma cells and efficiently inhibited the growth of subcutaneously injected tumors, even when treatment was initiated two weeks after tumor inoculation. An inhibitory affect of IL-12 on tumor growth, with a greater than two-fold increase in survival of inoculated animals, was also observed with the reticulum cell sarcoma M5076 and with the renal cell adenocarcinoma. In this latter tumor, complete remission, especially with peritumoral injection of IL-12, was observed in some animals; reinjection of the renca cells in the "cured" animals resulted in delayed growth of the tumor, suggesting that IL-12 may induce a memory immune response against the tumor" (paragraph spanning 4021-4022, references omitted).

The portion of Trinchieri cited is drawn to the work of Brunda et al (J. Exp. Med vol. 178, Oct 1993, p. 1223-1230). The Brunda et al study teaches that IL-12 has potent antitumor and anti-metastatic activities in several murine tumor models through an immune mediated T-cell dependent mechanism (p. 1228 column 2 last paragraph). However, Brunda et al teaches that future clinical trials with this cytokine will determine if the activity demonstrated in animals can be translated into efficacy against human malignancies (p. 1228 column 2 last paragraph). Neither Brunda et al or Trichineri et al discloses treatment with CpG oligonucleotides. In

addition, the instant claims are not limited to treatment in murine animals but include humans as well as other animals. See instant specification bottom of p. 11 and claim 37. Brunda et al teaches the differences in responses to IL-12 stimulation of human versus murine NK or T cells (see above) and teaches that more work needs to be done to determine if the activity demonstrated in animals can be translated into efficacy against human malignancies (p. 1228 column 2 last paragraph).

The review by Trinchieri et al also discloses the use of IL-12 in treatment of viral infections (see p. 4020 column 2 under virus infections). Trinchieri et al teaches that IL-12 is a potentiator of delayed type hypersensitivity and of cytotoxic lymphocyte responses and should be expected to play a role in the resistance against virus infections but however little information is available on either the importance of endogenous Il-12 or the effect of Il-12 treatment in viral infections. Trinchieri teaches that surprisingly Il-12 inhibited rather than enhanced CTL generation in Lympocytehoriomeningitis virus infected mice and the inhibitory effect was evident at particularly high doses of IL-12. While the instant oligonucleotide may stimulate production of IL-12 in a subject having a viral infection it is not clear how much IL-12 is produced and whether the amount produced in inhibits the CTL response to viral infection. The specification does not correlate the amount IL-12 production in response to the instant oligonucleotide with inhibition or enhancing CTL activity and thus one of skill in the art cannot predict based on the guidance in the specification and in the art how the IL-12 produced correlates with treatment of any type of viral infection especially in view of the fact that IL-12 in high doses inhibits the antiviral response to LCMV infection

Even though Applicants submit that IFN-gamma is produced from human T-lymphoblastoid line upon virus infection (Morris et al Infection and Immunity, 1982 35(2):533-536, p. 536 left column); and IFN-gamma has been identified as a key factor in immune responses to viral infections and that there is IFN-gamma production in response to influenza virus (Baumgarth et al. Journal of Virology, 1994, 68 (11):7575-7581); and that lymphokines, NK cells, Langerhans Cells are important in maintaining normal immunocompetence in the cervical mucosa and thus may play a role in treatment of viral infections including papillomavirus virus (Woodworth and Simpson. Am. J. Path., vol 142(5):1544-55, 1993; Schneider. Genitourin. Med., 1993, vol 69 (3): 165-73; Morris et al. Br J Obstet Gynecol, 1983,

vol 90(5):412-20); all these references do not disclose the induction of an immune response to CpG oligonucleotides and as evidenced by Trinchieri et al cytokine production does not predict efficacy for treatment of a viral infection and may be dependent on how much of the cytokine is produced. Treatment or elimination of HIV in HIV infected patients with the instant oligonucleotide is made more complex because of the impaired ability of HIV infected patients to produce IL-12 (Trinchieri et al p. 4020 to 4021 under IL-12 and HIV) which Applicants submit is important in treating viral infections. If a subject having HIV infection has an impaired IL-12 (important for stimulating NK cells which produce IFN gamma) producing ability then it is unpredictable based on Applicants disclosure and the teaching of the art that the instant oligonucleotide can treat or eliminate said HIV infection. Although, Trinchieri et al also teaches that studies in murine models suggest IL-12 may play a role in combating *Listeria monocytogenes* (an intracellular bacteria) infection (see p. 4018 column 2 under *Listeria monocytogenes*), the instant specification does not correlate the strength of the immune response generated by the instant oligonucleotide with treatment of *Listeria* or any other type of bacteria and studies in murine models with IL-12 may not predict efficacy in humans due to the differences in the responses to IL-12.

The state of the prior art provides evidence for the degree of predictability in the art and is related to the amount of direction or guidance needed in the specification as filed to meet the enablement requirement. The state of the prior art is also related to the need for working examples in the specification. Reasonable correlation must exist between the scope of the claims which is set forth above and the enablement set forth. The scope of the instant invention is very broad -treatment of any type of cancer, viral infection or bacterial infection in any type of subject. As set forth above, treating these conditions may not be predicted from just the ability of the instant oligonucleotide to induce cytokines or the other types of immune responses. The conditions are complex - cancer is complex, there are different types, and they respond differently by treatment (see for example, Hiyashi et al). Bacterial infections are complex to - different bacteria, intracellular or extracellular. Viral infections are complex and the physiology of the subject who has a viral infection may not respond to Il-12 (see for example Trichineri et al). The unpredictability of using murine models to predict efficacy in humans is also set forth supra. There are so many factors to be considered and mere induction/production of IL-12, IFN-

gamma, NK cell activation and IL-6 by the instant oligonucleotide does not reasonably correlate with the scope of the instant claims and it would require undue experimentation to practice the invention as claimed.

Status of Claims

Claims 28, 31-33, 35, 37, 39, 40 and 42-47 are rejected. No claims allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to OLUWATOSIN OGUNBIYI whose telephone number is 571-272-9939. The examiner can normally be reached on M-F 8:30 am- 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Robert Mondesi can be reached on 571-272-0956. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

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